New Information on Lead in Dirt and Dust as Related to the Childhood Lead Problem

by Gary Ter Haar* and Regine Aronow †

It has been known for many years that the eating of leaded paint is the prime cause of lead poisoning and elevated blood leads of children living in deteriorated housing. Recently, there has been speculation that children may eat dirt and dust contaminated with lead exhausted from cars and that this amount of ingested lead is sufficient to contribute significantly to the childhood lead problem.

This paper reports on a twopart study conducted to evaluate the validity of the dirt-and-dust hypotheses.

The first part of the study was made to determine the source of lead in dirt to which children are normally exposed. Dirt samples were taken in old urban areas around 18 painted frame houses and 18 houses of brick construction. Samples also were taken around seven old frame farmhouses remote from traffic. Based on the fact that lead concentrations in the dirt were similar in city and rural yards at corresponding distances from the houses, it is clear that nearly all of the lead in dirt around these houses is due to paint from the houses. Lead antiknock additives are therefore not a significant contributor to the lead content of dirt around houses where children usually play.

The second part of the study used a naturally occurring radioactive tracer ^{210}Pb to determine the relative amounts of dust and other lead-containing materials (e.g., paint) eaten by young children. This tracer is present in very low concentrations in paint and in significantly higher concentrations in fallout dust. Stable lead and ^{210}Pb were analyzed in fecal material from eight children suspected of having elevated body burdens of lead and ten children living in good housing where lead poisoning is not a problem.

The normal children averaged 4 μg Pb/g dry feces, with a range of 2 to 7. Of the eight children suspected of having elevated lead body burdens, two had fecal lead values within the normal range. However, the remaining six were 4 to 400 times as high. Despite these differences in fecal lead between the two groups, the groups were essentially identified in the ^{210}Pb content of their feces. The "elevated" children averaged 0.040 pCi of ^{210}Pb dry feces, while the normal group averaged 0.044 pCi/g. The results provide sound evidence that these children suspected of elevated lead body burden were not ingesting dust or air-suspended particulate.

Lead poisoning in children, especially those under 4 years old, is prevalent in areas of our larger cities where housing has deteriorated. Historically, almost all cases of lead poisoning in children have been attributed to eating paint chips with a high lead content (*1*, *2*). Recently, Fine et al. (*3*) in a study in Illinois, found that

*Ethyl Corporation, Research Laboratories, Detroit, Michigan 48220.

†Children's Hospital of Michigan, Detroit, Michigan 48904.

elevated blood lead was also common in smaller cities. In this study, they screened children from poor housing areas and concluded that the lead problem is not confined to large cities. The common factor between the large and small cities was deteriorated housing.

Recently, it has been speculated that dirt, dust, and air-suspended particulates may also contribute to lead poisoning in children (*4*). In order to consider these sources, it is necessary to define these terms. Air-suspended particulates are defined as solid particles in the air that can be removed by filtration. By dust we mean solid particles that settle from the air. Dust is usually measured by placing buckets or pans outside and allowing them to catch material that falls from the air. Equivalent terms used in this paper are dustfall and fallout dust. Dirt refers to dustfall mixed with other materials, including soil.

This paper considers two aspects of the childhood lead problem: (1) the major sources of lead in dirt around houses where children play; (2) the extent to which children take in dust and air-suspended particulates.

Lead in Dirt

Past studies have shown that deteriorating paint is an important source of elevated lead in dirt. Hardy et al. (*5*) have reported on analysis of lead in dirt near a barn remote from traffic in rural Lincoln, Massachusetts. Dirt next to the barn contained 2000 ppm of lead, and the level 20 ft from the barn was 160 ppm. Bertinuson and Clark (*6*) concluded that urban housing appears to be a larger contributor to elevated lead in dirt than emissions from vehicular exhaust. Fairey and Gray (*7*) found high concentrations of lead in dirt in yards, with the highest concentrations generally near the houses. They attributed this lead to paint and ashes.

In this study, we sampled dirt at nine sites around each of 18 frame houses in widely scattered urban areas of Detroit. These areas are characterized by old houses that had been painted with lead-based paint, presumably for many years. Analysis confirmed that all houses were coated with paint containing lead. Paint chips taken from the sides of the houses contained 3–26% lead. Most of the houses were vacant at the time of our sampling, but are in areas where the neighboring houses were occupied. Moreover, most of the vacant houses were in such a state that reoccupation is to be expected. Similarly, dirt samples were taken at nine sites around each of 18 houses of brick construction. In all cases, these houses have painted trim and were chosen so that the homes immediately adjacent were also brick to minimize contamination from the neighboring houses.

For each urban house, surface samples were taken at the following nine locations: (1) dirt in the street gutter; (2) dirt between the sidewalk and curb adjacent to the curb; (3) dirt two feet toward the house from the front sidewalk; (4)–(7) dirt on each of the four sides of the house within 2 ft of the house; (8), (9) dirt 10 ft from the house in the front and back yards.

We also sampled dirt around seven farmhouses in an area remote from traffic located about 30 miles from the nearest city and about 50 miles north of Detroit. All of these houses were on little-traveled gravel roads. Each house was set well back from the road, usually at least 150 ft. Samples were taken on all four sides of the farmhouses at 2, 10, and 20 ft from the houses. A sample was also taken on each farm several hundred feet away from the buildings and the road to allow a determination of the background level of lead. Samples were taken from the surface in the same manner as used for the urban samples.

Tables 1 and 2 show the data from this survey. Lead in dirt within 2 ft of the urban frame houses averaged 2010 ppm with no obvious bias for front, sides or back. Lead in dirt in the middle of the yards averaged 436 ppm, and again there was no bias toward front or back. The distribution around the brick houses was similar, but the lead

Table 1. Lead in dirt in Detroit.

		Lead in dirt, $\mu g/g$ dry dirt								
		Within 2 ft of house [a]				10 ft from house				
House type	House	Front	Back	Sides		Front	Back	Near sidewalk	Curb	Gutter
Painted frame	1	1919	3001	1170	748	985	351	449	660	596
	2	4121	674	7284	6003	536	289	1301	432	1079
	3	439	2193	1116	2548	278	608	326	610	508
	4	237	2539	1117	925	216	131	1482	680	n. d.
	5	920	2184	1211	1447	191	223	309	320	738
	6	126	233	186	916	58	157	343	321	1270
	7	n. d.	n. d.	1457	n. d.	n. d.	n. d.	627	404	645
	8	3420	n. d.	1380	5120	621	831	355	1957	1827
	9	179	372	611	1060	139	122	820	555	1047
	10	17590	4951	5552	7000	305	207	422	918	1387
	11	262	1585	5694	3402	197	219	506	338	1168
	12	295	292	140	104	170	149	152	220	n. d.
	13	556	246	446	254	229	285	266	328	1046
	14	1256	655	1206	4243	208	149	1958	331	550
	15	n. d.	162	1083	373	280	252	299	701	n. d.
	16	1077	1660	1894	1460	708	1220	425	419	935
	17	768	1094	220	1483	952	614	227	708	1277
	18	4068	3535	1452	1278	1530	1410	1017	400	415
	Average	2349	1586	2257	1846	447	425	627	572	966
Brick	19	380	222	106	146	77	72	246	301	564
	20	606	168	217	128	125	94	438	711	1670
	21	78	169	96	1540	103	48	1130	431	2085
	22	1030	701	838	725	148	188	485	881	1360
	23	352	344	883	486	203	480	416	966	3170
	24	687	197	91	222	219	97	263	303	656
	25	104	107	194	95	88	75	87	147	1070
	26	183	1915	474	1210	312	329	248	324	578
	27	382	835	597	4610 [b]	168	816	249	148	487
	28	377	283	1160	1500	228	163	403	2420	1410
	29	146	463	173	231	103	80	154	469	765
	30	146	203	102	269	108	84	169	330	600
	31	491	172	187	113	39	50	86	403	423
	32	140	72	1090	40	201	119	117	408	3140
	33	480	2350	800	632	316	417	301	395	867
	34	150	131	173	251	153	77	261	428	304
	35	366	442	218	281	111	244	317	750	2380
	36	227	243	276	2290	111	175	465	1210	298
	Average	351	501	426	595	156	200	324	612	1213

[a] Many of samples for painted frame house contained readily visible paint chips, especially house 10; n. d. denotes not determined.

[b] House next door was a painted frame house. This value not included in average.

levels were lower. The average concentration ranged from 351 to 595 ppm within 2 ft of the house and was 156 ppm in the front yard and 200 ppm in the back yard. As with the frame houses, there was no evidence of higher concentrations in the front yard compared to the back. Figure 1 is a graphical representation of the data.

The data for lead in dirt around the rural farmhouses (Table 2) are very similar to those for the urban frame houses (Table 1). The average lead concentration is 2529

Table 2. Lead in dirt in rural area: painted frame farmhouse.

House	Lead in dirt, μg/g dry dirt			
	2 ft from house [a]	10 ft from house [a]	20 ft from house [a]	Background
1	2162	417	67	9
2	450	429	144	27
3	6338	2093	166	26
4	1896	199	74	74
5	5184	556	640	12
6	840	428	107	63
7	831	141	268	94
Average	2529	609	209	44

[a] Each value is the average of four samples, one collected on each side of the house.

ppm within 2 ft of the farmhouses and 609 ppm at 10 ft and 209 ppm at 20 ft from the houses.

Table 3 shows the comparison between frame houses in the city, brick houses in the city, rural houses, and one literature source of lead around a barn. The comparison indicates that most of the lead in dirt is due to paint, based on the following reasoning. The lead in dirt within 2 ft of the frame houses in the city averages just over 2000 ppm. The lead in dirt 10 ft from these houses averages over 400 ppm and is similar in the front and back yard. If vehicular traffic were a significant source of lead, the front yard would contain more lead than the back. Since these data at 2 ft and 10 ft from the house are similar to our data from frame houses in rural areas and to the data of Hardy et al. (*5*), it is clear that traffic is not contributing significantly to lead in the dirt in the yards of the painted frame houses.

Table 3. Summary of lead in dirt.

	Lead in dirt, μg/g dry dirt	
	2 ft from house	10 ft from house
Frame houses, city	2010	436
Brick houses, city	468	178
Frame houses, rural	2529	609
Barn [a]	2000	570

[a] From Hardy et al. (*5*).

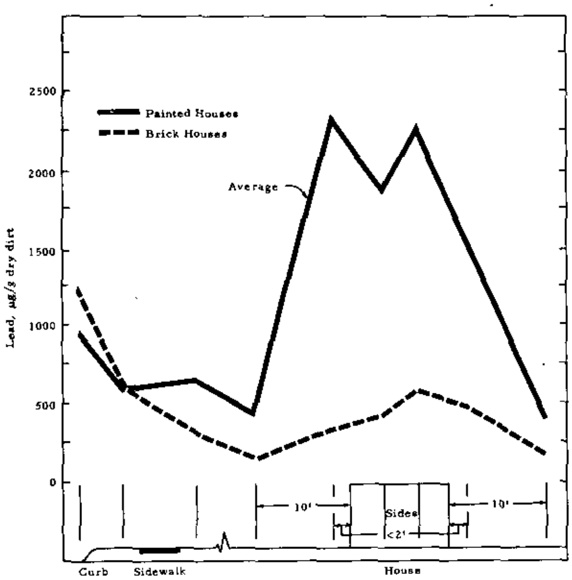

FIGURE 1. Lead in dirt around urban homes.

This conclusion is supported by the data on lead in dirt around the brick houses. The lead in dirt within 2 ft of these brick houses is more than double that at 10 ft, indicating that the painted trim of the house is the prime source. As expected, the much smaller painted surfaces of the brick houses result in much lower lead concentrations near these houses than near the painted frame houses. As with the painted frame houses, the concentrations 10 ft from the houses are similar in the front and back yards. Here again, it is evident that traffic does not have a significant effect. The lead in the street gutter was similar for both brick and frame houses. Thus, all evidence points to paint as the prime source of elevated lead in the yards, where the children would be most likely to play.

Children and Lead in Dust

The data from the first part of the study show that paint is the major source of lead in dirt around the houses where children usually play. The role of lead in air-suspended particles and in fallout dust must be considered separately, since lead in gasoline significantly contributes to lead contents of air-suspended particles and fallout dust (*8*).

To distinguish between the leaded paint a child might eat and the contribution of lead he might receive from eating dust, it is necessary to find a material that is present in dust but not in paint. The reverse would also be useful. Many elements in addition to lead are present in paint. These include titanium, zinc, calcium, barium, chromium, and aluminum, as well as traces of cobalt, manganese, and other metals. Unfortunately, these metals are also present in dust and dirt in relative amounts that are about the same as those in paint.

Lead-210, a naturally occurring radioactive isotope of lead, is a useful tracer for this purpose. It is generated from radon, which is present in the soil. Part of the radon escapes to the air and part stays in the soil. The radon disintegrates with a half-life of 3 days to produce ^{210}Pb, which has a half-life of 22 yr (*9*). Because it is present in the atmosphere, fallout dust is enriched in ^{210}Pb, while paint has very low concentrations of ^{210}Pb.

We have analyzed samples of paint, dust, dirt, and air-suspended particulate for their ^{210}Pb content. Table 4 shows the resulting ranges of the ^{210}Pb content of these materials.

Table 4. ^{210}Pb in nonfood materials.

Material	^{210}Pb, pCi/g
Paint chips	0.005–0.07
Urban airborne particulate	60–150
Fallout dust	3–30
Vacuum-cleaner sweepings	0.4–1
Yard dirt	0.3–2
Street dirt	0.4–4

The large differences in concentration occur because of the vast difference in the amount of solids associated with the ^{210}Pb, not because the ^{210}Pb varies so greatly. For example, the amount of dust in the air per cubic meter is very small, and the amount of solids in a handful of dirt is enormous in comparison.

The concept was to use ^{210}Pb as a tracer to determine the amount of dust and perhaps the amount of dirt eaten daily by a child. As lead and ^{210}Pb are absorbed poorly in the gut, an estimate of the lead and lead-210 can be made from analyses of fecal matter. If a child has a high level of lead in his fecal matter and a normal level of ^{210}Pb, we would conclude that the lead elevation is a result of eating paint. However, if both the lead and lead-210 are high in the fecal matter, we would conclude that dust and dirt are contributors in addition to paint.

At Children's Hospital of Michigan, we collected urine and fecal samples from children who were suspected of having elevated body burdens of lead. The evidence used was one or all of the following: (1) x-ray showed radioopaque materials in the gut; (2) history of pica; (3) elevated blood lead; (4) x-ray showed lead lines in the long bones. Fecal and urine samples were taken from eight such children. These children were 1 to 3 years old and all had exhibited pica tendencies. All stool and urine were separately collected during the first 24 hr after admission to the hospital to insure samples representative of the child's usual environment, not that of the hospital. To provide a baseline, combined stool and urine samples were taken from 10 children of the same age level (1–3 yr) who lived in good housing in Detroit and its suburbs where lead poisoning is not a problem. All samples were collected during the late spring and early summer months. These samples were analyzed for stable lead and ^{210}Pb.

Table 5 shows the lead and ^{210}Pb data for the normal children and the children with pica. The normal children average 4 μgPb/g dry feces, with a range of 2 to 7. Of the eight children suspected of having elevated lead body burdens, two had fecal lead values (4 and 7 μg lead) within the normal range.

Table 5. Lead and Lead-210 in excreta.

Normal children		Hospitalized children	
Stable Pb, μg/g dry	^{210}Pb, pCi/g dry	Stable Pb, μg/g dry	^{210}Pb, pCi/g dry
3	0.019	19	0.046
2	0.021	20	0.018
3	0.027	18	0.024
7	0.120	49	0.047
7	0.087	4	0.050
3	0.041	7	0.039
3	0.026	40	0.063
5	0.028	1640	0.037
4	0.044		
4	0.024		
Avg.	0.044	Avg.	0.040

However, the remaining six were 4 to 400 times higher. Despite these differences in fecal lead between the two groups, the groups were essentially identical in the ^{210}Pb content of their feces. The "elevated" children averaged 0.040 pCi of ^{210}Pb per gram dry feces, while the normal group averaged 0.044 pCi/g.

Statistical examination of the ^{210}Pb data show that they are lognormally distributed and that there is no statistical difference in the concentration of ^{210}Pb between the two groups. The results of this experiment do not support the hypothesis that young children with pica eat dust.

The child who excreted 1640 μg Pb/g dry feces was treated with chelating agents to lower the blood lead (108 μg Pb/100 ml blood). Table 6 shows the data for stable lead and lead-210 in the urine before and during treatment. As shown, lead output in the urine rose sharply during the treatment, while ^{210}Pb output varied hardly at all. This shows not only that this child was not eating dust and dirt on the day we examined his feces, but also that he had not been doing so earlier. If he had been eating materials high in ^{210}Pb, his tissue levels would have been elevated and ^{210}Pb would have increased in the urine along with the stable lead. This did not happen.

The ^{210}Pb data for these 18 children can be related to the amount of lead-210 that is present normally in the diet. The normal children in this study excreted an average of 0.67 pCi of ^{210}Pb per day in 15 g excreta (dry weight). This value agrees very well with an estimation based on ^{210}Pb data of Morse and Welford (*10*) for adults. They found that adults ingested about 1.4 pCi of ^{210}Pb per day. On the basis of literature estimates that a child consumes about half the food of an adult, (*11, 12*), an intake of 0.7 pCi of ^{210}Pb per day would be expected for a child.

Table 6. Lead and ^{210}Pb in urine.

Collection	Stable Pb, μg/l.	^{210}Pb, pCi/l.
1	147	0.23
2	153	0.28
3	121	0.74
	Treatment with D-Penicillamine (oral) began	
4	3820	0.27
5	1960	0.18
6	2030	0.42
7	6140	a
8	1400	0.27
9	2880	0.51
10	1170	0.48
11	3210	a
12	2530	a
13	1125	a
14	1780	0.40
	Treatment began with calcium disodium ethylenediaminetetraacetic acid and 2,3-dimercaptolpropanol (muscularly)	
15	4180	0.55

[a] Sample was partly used in the hospital, leaving too small an amount for ^{210}Pb determination.

Study Techniques

Soil Samples

Soil samples were collected by taking the topmost layer of soil. All soil samples were dried at 100°C overnight. Lead was extracted with hot dilute nitric acid and determined by atomic absorption.

Biological Samples

The samples from the normal children were collected in acid-washed plastic containers for 24 hours by the mother according to the following instructions.

1. Collect all urine and feces for 24 hr and place in container provided.

2. If possible, child should use training chair. Samples are more easily handled in this manner than with diapers. A small amount of the (distilled) water provided should be used to wash out receiver. This water should be added to container provided.

3. If child is still in diapers, use disposable diapers. Place entire disposable portion in container provided. Keep the sample separated from any samples collected using training chair.

4. If child should have an accident and sample cannot be collected, the entire day's collection should be discarded and collection begun again the next day for 24 hr.

Urine and fecal samples from children admitted to Children's Hospital were collected separately in lead-free containers during the first 24 hr after admission.

All fecal and urine samples were weighed and dried at 100°C. The dry weight was recorded and the sample was taken into solution with nitric and perchloric acid. The lead was taken into methyl isobutyl ketone and analyzed by atomic absorption.

^{210}Pb Analysis

Only a small portion of the methyl isobutyl ketone–lead solution was used to determine lead. The remainder was oxidized with nitric acid. After fuming three times with a few milliliters of HCl, the ^{210}Pb was determined by the method of Black (*13*).

Summary

This report has described the results of a two-part study to determine whether lead emitted from motor vehicles contributes to the lead problem in small children. In the first part, we determined lead in dirt around houses in urban areas and rural areas. The data from the urban areas clearly show that the principal cause of elevated lead in the dirt in the yards is leaded paint on these houses. These data were confirmed by measurements of lead in dirt around farmhouses. The distribution was the same as that in the city, showing traffic was not an important consideration.

In the second part of the study, we determined the amount of air-suspended particulate or dustfall a child might eat. We used a naturally occurring tracer, ^{210}Pb, which is present in relatively large amounts in dust but nearly absent from paint. The results showed that children with pica (and other evidence of high lead intake) and normal children excreted identical amounts of ^{210}Pb. Consequently, dust and air-suspended particulate were not the sources of lead in these urban children.

REFERENCES

1. Chisolm, J. J., Jr., Lead poisoning. Sci. American 224: 15 (1971).
2. Chisolm, J. J., Jr., Childhood lead intoxication, diagnosis, management and prevention. *Med. Times* 98: 92 (1970).
3. Fine, P. R., et al. Pediatric blood lead levels. JAMA 221: 1475 (1972).
4. U.S. Environmental Protection Agency. EPA's position on the health effects of airborne lead. Health Effects Branch, Processes and Effects Division, Office of Research and Monitoring, U.S. Environmental Protection Agency, Washington, D.C., November 29, 1972.
5. Hardy, H. L., et al. Lead as an environmental poison, Clin. Pharmacol. Therap. 12: 982 (1971).
6. Bertinuson, J. R., and Clark, C. S. The contribution to lead content of soils from urban housing. Interface, 6: 1073 (1973).
7. Fairey, F. S., and Gray, J. W., III. Soil lead and pediatric lead poisoning in Charleston, S.C. J.S.C. Med. Assoc.: 79 (1970).
8. National Academy of Sciences—National Research Council, Airborne Lead in Perspective. National Academy of Sciences, Washington, D.C., 1972.
9. Patterson, R. L., and Lockhart, L. B., Jr. In: The Natural Radiation Environment, J. Adams and W. Louden, Eds., The University of Chicago Press, Chicago, 1964, pp. 383–392.
10. Morse, R. S., and Welford, G. A. Dietary intake of ^{210}Pb. Health Phys. 21: 53 (1971).
11. King, B. C. Maximum daily intake of lead without excessive body lead-burden in children. Amer. J. Dis. Child. 122: 337 (1971).
12. Barltrop, D. Sources and Significance of Environmental Lead for Children. Paper presented at International Symposium, Environmental Health Aspects of Lead, Amsterdam, October 2–6, 1972.
13. Black, S. C. Low-level polonium and radiolead analysis. Health Phys. 7: 87 (1961).